

	Type	L #	Hits	S arch Text	DBs	Time Stamp	Com men ts	Err or Def ini tio n	Er ro rs
1	BRS	L1	4	oxygen adj labile adj species	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 18:41		0	
2	BRS	L2	53	oxygen adj labile	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 18:42		0	
3	BRS	L3	4561	retinoid	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 18:53		0	
4	BRS	L4	1168	N-acetylcysteine	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 18:45		0	
5	BRS	L5	56	3 same 4	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 18:45		0	
6	BRS	L6	52350	choleciferol or (vitamin adj K) or tocopherol or (ascorbic adj acid)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 18:46		0	
7	BRS	L7	4	5 same 6	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 18:47		0	
8	BRS	L8	1	N-acetylcysteine same retinoid same (ascorbic adj acid) same tocopherol	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 18:51		0	
9	BRS	L9	9307	retinoid or retinol or retinamide or (retinoic adj acid)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 18:58		0	

	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
10	BRS	L10	65	9 same 4	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 18:54			0
11	BRS	L11	10	10 same 6	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 18:55			0
12	BRS	L12	19211 4	niacin or thiamine or riboflavin or (folic adj acid) or pyroxidine or (pantothenic adj acid) or niacinamide or (lipoic adj acid) or (dihydrolipoic adj acid) or (amino adj acid)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 19:00			0
13	BRS	L13	2	11 same 12	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3. 19:00			0
14	BRS	L14	58518 2	humectant or antioxidant or preservative or fragrance or (surface adj active adj agent) or binder or (skin adj protectant adj agent)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 19:06			0
15	BRS	L15	4	11 same 14	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 19:07			0
16	BRS	L16	5	10 same (ascorbic adj acid)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/01/2 3 19:07			0

> d his

(FILE 'HOME' ENTERED AT 19:11:34 ON 23 JAN 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA'
ENTERED AT

19:11:57 ON 23 JAN 2003

L1 165660 S RETINOID OR RETINOL OR RETINAMIDE OR (RETINOIC ACID)
L2 17009 S N-ACETYLCYSTEINE
L3 273934 S CHOLESCIFEROL OR (VITAMIN K) OR TOCOPHEROL OR
(ASCORBIC ACID)
L4 119 S L1 (P) L2
L5 696 S L2 (P) L3
L6 19 S L4 (P) L3
L7 11 DUPLICATE REMOVE L6 (8 DUPLICATES REMOVED)
L8 154568 S NIACIN OR THIAMINE OR RIBOFLAVIN OR (FOLIC ACID) OR
PYRODOXIN
L9 1958819 S (LIPOIC ACID) OR (DIHYDROLIPOIC ACID) OR (AMINO ACID)
L10 2103109 S L8 OR L9
L11 3 S L7 (P) L10
L12 1 S L7 (P) COMPOSITION
L13 552432 S HUMECTANT OR ANTIOXIDANT OR PRESERVATIVE OR
FRAGRANCE OR (SU
L14 2 S L7 AND L13

=> log y

FILE 'HOME' ENTERED AT 19:11:34 ON 23 JAN 2003

=> file medline caplus biosis embase scisearch agricola		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 19:11:57 ON 23 JAN 2003

FILE 'CAPLUS' ENTERED AT 19:11:57 ON 23 JAN 2003
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FILE 'EMBASE' ENTERED AT 19:11:57 ON 23 JAN 2003
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FILE 'AGRICOLA' ENTERED AT 19:11:57 ON 23 JAN 2003

=> s retinoid or retinol or retinamide or (retinoic acid)
L1 165660 RETINOID OR RETINOL OR RETINAMIDE OR (RETINOIC ACID)

=> s N-acetylcysteine
L2 17009 N-ACETYLCYSTEINE

=> s cholesciferol or (vitamin K) or tocopherol or (ascorbic acid)
L3 273934 CHOLESCIFEROL OR (VITAMIN K) OR TOCOPHEROL OR (ASCORBIC ACID)

=> s l1 9p) l2
MISSING OPERATOR L1 9P
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l1 (p) l2
L4 119 L1 (P) L2

=> s l2 (p) l3
L5 696 L2 (P) L3

=> s l4 (p) l3
L6 19 L4 (P) L3

=> duplicate remove l6
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETED FOR L6
L7 11 DUPLICATE REMOVE L6 (8 DUPLICATES REMOVED)

=> d l7 1-11 ibib abs

L7 ANSWER 1 OF 11 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 1
ACCESSION NUMBER: 2002:162784 CAPLUS
DOCUMENT NUMBER: 137:194801
TITLE: Cancer chemoprevention - present and future
AUTHOR(S): Fujiki, Hirota; Suganuma, Masami
CORPORATE SOURCE: Saitama Cancer Center, Japan
SOURCE: Biotherapy (Tokyo, Japan) (2002), 16(1), 1-9
CODEN: BITPE9; ISSN: 0914-2223
PUBLISHER: Gan to Kagaku Ryohosha
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review with refs. The term "cancer chemoprevention", defined as
prevention of the occurrence of cancer by administration of one or more

compds., was coined by Michael B. Sporn in 1976. The significance of cancer chemoprevention is now internationally accepted, and interest in Japan is accelerating. This article looks at notable results of clinical trials conducted in the U.S. and Europe, and touches upon research activities in Japan. The good news includes promising results on cancer prevention in the breast, colon and liver. Main topics: 1) Breast cancer prevention trials with tamoxifen have provided effective results for individuals in the high risk group, patients with premalignant lesions-ductal carcinoma in situ (DCIS) - and contralateral breast cancer patients. 2) Primary lung cancer prevention trials with alpha-***tocopherol***, .beta.-carotene (ATBC) in Finland, and with .beta.-carotene and ***retinol*** (CARET) in the U.S., showed an unexpected increase in lung cancer incidence, and recently reported results of a EUROSCAN study did not show any benefits from vitamin A and ***N*** - ***acetylcysteine***. These three major studies indicate that a new approach is required. 3) Encouraging results with prostate cancer prevention by finasteride are anticipated. 4) The FDA has approved celecoxib, a selective Cox-2 inhibitor, for the prevention of polyp development in patients with familial adenomatous polyposis. 5) Acyclic ***retinoid***, polyphenolic acid prevented second primary hepatomas after surgical resection of the original tumor or percutaneous injection of ethanol, mediated through clonal deletion of malignant cells in the remnant liver. 6) Finally, we discuss cancer prevention with green tea. Based on results of basic studies of (-)-epigallocatechin gallate (EGCG) and green tea polyphenols, and also results of a prospective cohort study with 8,552 individuals, we are now moving toward prevention of cancer in various organs (both primary tumor and recurrence) by introducing the "Saitama System": the equiv. of 10 Japanese-size cups of green tea per day (2.5 g green tea ext. per day) in a combination of daily beverage and green tea tablets. It is our hope that this article will provide information that will spur an increase in cancer prevention trials in Japan.

L7 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2001:649589 CAPLUS
DOCUMENT NUMBER: 136:67768
TITLE: Lung cancer
AUTHOR(S): Worden, Francis P.; Kalemkerian, Gregory P.
CORPORATE SOURCE: Department of Medicine, University of Michigan, Ann Arbor, MI, 48109, USA
SOURCE: Cancer Treatment and Research (2001), 106(Cancer Chemoprevention), 183-219
CODEN: CTRREP; ISSN: 0927-3042
PUBLISHER: Kluwer Academic Publishers
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Lung cancer is the result of the accumulation of numerous genetic and epigenetic defects within bronchial epithelial cells. Clinical trials demonstrated that chemopreventive agents, such as .beta.-carotene, ***retinol*** /retinyl palmitate, 13-cis- ***retinoic*** ***acid***, .alpha.- ***tocopherol***, and ***N*** - ***acetylcysteine*** were ineffective in preventing lung cancer in select high-risk populations, and .beta.-carotene may even be harmful in active smokers. Increasing knowledge of the molecular events involved in lung carcinogenesis have led to the development of many strategies that specifically target derangements in signal transduction or cell cycle regulatory pathways.

REFERENCE COUNT: 189 THERE ARE 189 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE REFORMAT

L7 ANSWER 3 OF 11

MEDLINE

DUPLICATE 2

ACCESSION NUMBER: 2001513114 MEDLINE
DOCUMENT NUMBER: 21445113 PubMed ID: 11560978
TITLE: Lung cancer chemoprevention: an integrated approach.
AUTHOR: Lippman S M; Spitz M R
CORPORATE SOURCE: Departments of Clinical Cancer Prevention, Thoracic/Head and Neck Medical Oncology, and Epidemiology, University of Texas M.D. Anderson Cancer Center, Houston, TX 77030, USA..
slippman@mdanderson.org
CONTRACT NUMBER: CA16672 (NCI)
CA45809 (NCI)
CA55769 (NCI)

SOURCE: JOURNAL OF CLINICAL ONCOLOGY, (2001 Sep 15) 19 Suppl)
 74S-82S. Ref: 87
 Journal code: 8309333. ISSN: 0732-183X.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200110
 ENTRY DATE: Entered STN: 20010919
 Last Updated on STN: 20011015
 Entered Medline: 20011011

AB Lung cancer is the leading cause of cancer deaths in the United States and the world, with grim incidence and mortality figures underscoring the need for new approaches, such as chemoprevention, for controlling this disease. There have been definitive, randomized, controlled lung-cancer chemoprevention trials in the three chemoprevention trial settings: primary (healthy high-risk [eg, smokers]), secondary (pre-malignant lesions), and tertiary (prevention of second primary tumors in previously treated patients), all of which produced negative (either neutral or harmful) primary end point results. These trials established that lung cancer was not prevented by alpha- ***tocopherol***, beta-carotene, ***retinol***, retinyl palmitate, ***N*** - ***acetylcysteine***, or isotretinoin in smokers. Provocative leads of the definitive trials include the possible activity of isotretinoin in never and former smokers and that of alpha- ***tocopherol*** in prostate cancer prevention. A major area of lung cancer research is molecular epidemiologic study of highest smoking-related risk based on the interactions between tobacco carcinogens, genetic polymorphisms involved in activating and detoxifying these carcinogens, and host-cell efficiency in monitoring and repairing tobacco carcinogen-DNA damage. The future of lung cancer chemoprevention will rely heavily on molecular studies of carcinogenesis and drug mechanisms to develop novel chemopreventive targets and drugs, risk markers, and surrogate end point biomarkers; new preclinical drug-testing models; novel imaging techniques for monitoring agent activity; and molecular epidemiologic risk models for identifying the highest-risk current and former smokers.

L7 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2001:746946 CAPLUS
 DOCUMENT NUMBER: 136:47875
 TITLE: Lung cancer chemoprevention: an integrated approach
 AUTHOR(S): Lippman, Scott M.; Spitz, Margaret R.
 CORPORATE SOURCE: Departments of Clinical Cancer Prevention, University of Texas M.D. Anderson Cancer Center, Houston, TX, 77030, USA
 SOURCE: Journal of Clinical Oncology (2001), 19(18, Suppl.), 74s-82s
 CODEN: JCONDN; ISSN: 0732-183X
 PUBLISHER: Lippincott Williams & Wilkins
 DOCUMENT TYPE: Journal; General Review
 LANGUAGE: English

AB A review. Lung cancer is the leading cause of cancer deaths in the United States and the world, with grim incidence and mortality figures underscoring the need for new approaches, such as chemoprevention, for controlling this disease. There have been definitive, randomized, controlled lung-cancer chemoprevention trials in the three chemoprevention trial settings: primary (healthy high-risk [eg, smokers]), secondary (pre-malignant lesions), and tertiary (prevention of second primary tumors in previously treated patients), all of which produced neg. (either neutral or harmful) primary end point results. These trials established that lung cancer was not prevented by alpha- ***tocopherol***, beta-carotene, ***retinol***, retinyl palmitate, ***N*** - ***acetylcysteine***, or isotretinoin in smokers. Provocative leads of the definitive trials include the possible activity of isotretinoin in never and former smokers and that of alpha- ***tocopherol*** in prostate cancer prevention. A major area of lung cancer research is mol. epidemiol. study of highest smoking-related risk based on the interactions between tobacco carcinogens, genetic polymorphisms involved in activating and detoxifying these carcinogens, and host-cell efficiency in monitoring

and repairing tobacco carcinogen-DNA damage. The future of lung cancer chemoprevention will rely heavily on mol. studies of carcinogenesis and drug mechanisms to develop novel chemopreventive targets and drugs, risk markers, and surrogate end point biomarkers; new preclin. drug-testing models; novel imaging techniques for monitoring agent activity; and mol. epidemiol. risk models for identifying the highest-risk current and former smokers.

REFERENCE COUNT: 87 THERE ARE 87 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2001:482634 CAPLUS

DOCUMENT NUMBER: 135:298715

TITLE: Effects of antioxidant vitamins on anti-IgE-induced mediator release from human basophils

AUTHOR(S): Strenze, N.; Grabbe, J.; Plath, K. E. S.; Wolff, H. H.; Gibbs, B. F.

CORPORATE SOURCE: Department of Dermatology, Medical University of Lubeck, Lubeck, D-23538, Germany

SOURCE: Inflammation Research (2001), 50(Suppl. 2), S49-S50
CODEN: INREFB; ISSN: 1023-3830

PUBLISHER: Birkhaeuser Verlag

DOCUMENT TYPE: Journal

LANGUAGE: English

AB It was examd. whether antioxidants may provide beneficial effects by inhibiting IgE-dependent basophil mediator release. ***Ascorbic***
acid, ***N*** - ***acetylcysteine*** and glutathione were dissolved in RPMI 1640 medium and the pH was cor. to 7.4. Glutathione and its precursor ***N*** - ***acetylcysteine*** had no influence on IgE-dependent histamine release from human basophils, while ambroxol was shown to be a highly efficacious inhibitor. All the vitamins tested showed no effect on histamine release in the absence of anti-IgE. In the histamine release expts., ambroxol was the only effective inhibitor of interleukin (IL)-4 and IL-13 releases. Compared to solvent controls, ***N*** - ***acetylcysteine***, ***retinoic*** ***acid*** and .beta.-carotene did not considerably alter anti-IgE induced cytokine releases. ***Ascorbic*** ***acid*** strongly enhanced IL-13 release, which was not significant because of the donor variation in IL-13 release. A major role for reactive oxygen species in the IgE-dependent activation of human basophils was not supported by the resulting data.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2000:839120 CAPLUS

DOCUMENT NUMBER: 134:21446

TITLE: Compositions for stabilizing oxygen-labile pharmaceuticals

INVENTOR(S): Kung, John; Liu, Jue-chen

PATENT ASSIGNEE(S): Johnson & Johnson Consumer Companies, Inc., USA

SOURCE: Eur. Pat. Appl., 17 pp.

CODEN: EPXXDW

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1055720	A2	20001129	EP 2000-304519	20000526
EP 1055720	A3	20010307		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
CA 2309520	AA	20001128	CA 2000-2309520	20000525
JP 2001011441	A2	20010116	JP 2000-158635	20000529
CN 1284327	A	20010221	CN 2000-118833	20000529
BR 2000003780	A	20010403	BR 2000-3780	20000616
US 2002123460	A1	20020905	US 2001-33492	20011227

PRIORITY APPLN. INFO.: US 1999-136442P P 19990528
US 1999-361425 A 19990727

AB This invention relates to compns. and methods for stabilizing oxygen-labile species. More particularly, it relates to compns. contg. 1

TITLE: Dehydroepiandrosterone synergizes with
 antioxidant supplements for immune restoration
 in old as well as retrovirus-infected mice
 AUTHOR(S): Jiang, Shuguang; Lee, Jeongmin; Zhang, Zhen; Inserra,
 Paula; Solkoff, David; Watson, Ronald R.
 CORPORATE SOURCE: Arizona Prevention Center, Univ. Arizona, Tucson, AZ,
 85724, USA
 SOURCE: Journal of Nutritional Biochemistry (1998), 9(7),
 362-369
 CODEN: JNBIEL; ISSN: 0955-2863
 PUBLISHER: Elsevier Science Inc.
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB Prod. of the ***antioxidant*** hormone dehydroepiandrosterone (DHEA) declines as immunosenescence develops in the elderly. Very old C57BL/6 female mice (29 mo), survivors after 71% had died due to aging, were evaluated after DHEA sulfate 0.01% addn. to the drinking water for 6 wk. DHEA increased the T-cell proliferation, restored secretion of Th1 cytokines (interleukin IL-2), decreased interferon-.gamma. (IFN-.gamma.) prodn., and normalized (lowered) the Th2 cytokine (IL-4 and IL-6) secretion. The survival was increased in the 29-mo-old mice treated by DHEA. DHEA sulfate, the storage form of DHEA, decreased the immune dysfunctions caused by increased oxidn. during LP-BM5 murine leukemia retrovirus infection. To verify the synergistic effect of DHEA + ***antioxidant*** nutrients, 17-mo-old mice were fed with ***antioxidants*** (mixt. of coenzyme Q10, d-.alpha.- ***tocopherol***, L- ***ascorbic***, ***acid***, L-carnitine, ***N***, ***acetylcysteine***, ***retinol***, Se, Mg, Zn) or ***antioxidants*** + DHEA sulfate for 16 wk. DHEA sulfate + ***antioxidants*** increased the B-cell proliferation and IL-2 secretion and maintained the Th2 cytokine secretion and hepatic vitamin E levels close to the levels seen in old noninfected mice than did ***antioxidant*** supplementation alone. Thus, DHEA alone, and esp. DHEA sulfate plus ***antioxidant*** nutrients, can prevent immune dysfunctions in very old and in old retrovirus-infected mice.

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 19:11:34 ON 23 JAN 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 19:11:57 ON 23 JAN 2003

L1 165660 S RETINOID OR RETINOL OR RETINAMIDE OR (RETINOIC ACID)
 L2 17009 S N-ACETYLCYSTEINE
 L3 273934 S CHOLESCIFEROL OR (VITAMIN K) OR TOCOPHEROL OR (ASCORBIC ACID)
 L4 119 S L1 (P) L2
 L5 696 S L2 (P) L3
 L6 19 S L4 (P) L3
 L7 11 DUPLICATE REMOVE L6 (8 DUPLICATES REMOVED)
 L8 154568 S NIACIN OR THIAMINE OR RIBOFLAVIN OR (FOLIC ACID) OR PYRODOXIN
 L9 1958819 S (LIPOIC ACID) OR (DIHYDROLIPOIC ACID) OR (AMINO ACID)
 L10 2103109 S L8 OR L9
 L11 3 S L7 (P) L10
 L12 1 S L7 (P) COMPOSITION
 L13 552432 S HUMECTANT OR ANTIOXIDANT OR PRESERVATIVE OR FRAGRANCE OR (SU
 L14 2 S L7 AND L13

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	113.84	114.05

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-5.86	-5.86

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or more oil- and/or water-soluble oxygen-labile species and one or more stabilizing elements. It also relates to methods of making such compns. and methods of using such compns. Thus, a formulation contained water 73.96, disodium EDTA 0.20, phenoxyethanol 0.73, methylparaben 0.20, propylparaben 0.07 and hydroxyethyl cellulose 1.00% for the water phase; BHT 0.10, GMS 2.00 cetearyl glucoside 3.000, C12-15 alkyl benzoate 2.00, avobenzone 2.00, octyl methoxycinnamate 4.00, and ascorbyl palmitate 0.50% for the oil phase; ***ascorbic*** 5.00, ***acid*** 0.05, ***tocopherol*** 0.05, ***retinol*** 0.25, lactoferrin and thioxanthine and uric acid 1.00, ***N*** - ***acetylcysteine*** 0.01, EtOH 2.78 and 20% NaOH 9.04% as the additives. After a 13-wk incubation at 40.degree., 90% vitamin C and 96% vitamin A remained in the compn.

L7 ANSWER 7 OF 11 MEDLINE DUPLICATE 3
 ACCESSION NUMBER: 2000126354 MEDLINE
 DOCUMENT NUMBER: 20126354 PubMed ID: 10657911
 TITLE: Lung cancer chemoprevention.
 AUTHOR: Khuri F R; Lippman S M
 CORPORATE SOURCE: Departments of Thoracic/Head and Neck Medical Oncology, The University of Texas M. D. Anderson Cancer Center, Houston, Texas 77030, USA.
 SOURCE: SEMINARS IN SURGICAL ONCOLOGY, (2000 Mar) 18 (2) 100-5.
 Ref: 46
 Journal code: 8503713. ISSN: 8756-0437.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200003
 ENTRY DATE: Entered STN: 20000320
 Last Updated on STN: 20000320
 Entered Medline: 20000307

AB Lung cancer is the leading cause of cancer death in the United States. The persisting grim lung cancer incidence and mortality figures argue powerfully for new approaches such as chemoprevention for controlling this disease. ***Retinoids*** are among the most intensively studied cancer chemoprevention agents, including in the lung. Several randomized clinical or translational chemoprevention trials (e.g., of ***retinoids***, beta-carotene, or combined folic acid and vitamin B(12)) have been conducted in lung pre-malignancy. ***Retinoid*** studies have produced important data on molecular/cellular markers of lung carcinogenesis, e.g., loss of heterozygosity (LOH) at 3p and 9p and ***retinoic*** ***acid*** receptor-beta (RAR-beta). Two large randomized trials with a lung cancer endpoint, the Alpha- ***Tocopherol***, Beta-Carotene (ATBC) Prevention Study and the Beta-Carotene and ***Retinol*** Efficacy Trial (CARET), found that beta-carotene (+/- ***retinol***) was harmful (in smokers). Recently completed lung-second-primary-tumor-prevention trials include the ***retinoids*** retinyl palmitate and 13-cis- ***retinoic*** ***acid*** (13cRA) and ***N*** - ***acetylcysteine*** (NAC). Vitamin E and selenium show promise for lung cancer prevention, based on positive secondary/subset analyses of three large-scale, randomized National Cancer Institute (NCI) cancer prevention trials. Future directions of lung cancer chemoprevention include the study of molecular markers of risk and drug activity, molecular targeting study, improved imaging techniques (e.g., molecular imaging) and new drug delivery systems.
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L7 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2003 ACS
 ACCESSION NUMBER: 1998:497254 CAPLUS
 DOCUMENT NUMBER: 129:230153
 TITLE: Dehydroepiandrosterone synergizes with antioxidant supplements for immune restoration in old as well as retrovirus-infected mice
 AUTHOR(S): Jiang, Shuguang; Lee, Jeongmin; Zhang, Zhen; Inserra, Paula; Solkoff, David; Watson, Ronald R.
 CORPORATE SOURCE: Arizona Prevention Center, Univ. Arizona, Tucson, AZ, 85724, USA
 SOURCE: Journal of Nutritional Biochemistry (1998), 9(7),

362-369
CODEN: JNL; ISSN: 0955-2863
Elsevier Science Inc.

PUBLISHER:
DOCUMENT TYPE:
LANGUAGE:

Journal
English

AB Prodn. of the antioxidant hormone dehydroepiandrosterone (DHEA) declines as immunosenescence develops in the elderly. Very old C57BL/6 female mice (29 mo), survivors after 71% had died due to aging, were evaluated after DHEA sulfate 0.01% addn. to the drinking water for 6 wk. DHEA increased the T-cell proliferation, restored secretion of Th1 cytokines (interleukin IL-2), decreased interferon- γ . (IFN- γ .) prodn., and normalized (lowered) the Th2 cytokine (IL-4 and IL-6) secretion. The survival was increased in the 29-mo-old mice treated by DHEA. DHEA sulfate, the storage form of DHEA, decreased the immune dysfunctions caused by increased oxidn. during LP-BM5 murine leukemia retrovirus infection. To verify the synergistic effect of DHEA + antioxidant nutrients, 17-mo-old mice were fed with antioxidants (mixt. of coenzyme Q10, d- α -

tocopherol, L- ***ascorbic***, ***acid***, L-carnitine, ***N*** - ***acetylcysteine***, ***retinol***, Se, Mg, Zn) or antioxidants + DHEA sulfate for 16 wk. DHEA sulfate + antioxidants increased the B-cell proliferation and IL-2 secretion and maintained the Th2 cytokine secretion and hepatic vitamin E levels close to the levels seen in old noninfected mice than did antioxidant supplementation alone. Thus, DHEA alone, and esp. DHEA sulfate plus antioxidant nutrients, can prevent immune dysfunctions in very old and in old retrovirus-infected mice.

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 9 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:55925 BIOSIS
DOCUMENT NUMBER: PREV199800055925
TITLE: Chemoprevention of aerodigestive cancer.
AUTHOR(S): Berwick, Marianne (1); Schantz, Stimson
CORPORATE SOURCE: (1) Dep. Epidemiology Biostatistics, Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021 USA
SOURCE: Cancer and Metastasis Reviews, (1997) Vol. 16, No. 3, pp. 329-347.
ISSN: 0167-7659.
DOCUMENT TYPE: General Review
LANGUAGE: English

L7 ANSWER 10 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1993:421810 BIOSIS
DOCUMENT NUMBER: PREV199345069435
TITLE: Cancer prevention research trials.
AUTHOR(S): Greenwald, Peter (1); Malone, Winfred F. (1); Cerny, Mary E.; Stern, Harriet R.
CORPORATE SOURCE: (1) Div. Cancer Prevention and Control, Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20892 USA
SOURCE: Vande Woude, G. F. [Editor]; Klein, G. [Editor]. Advances in Cancer Research, (1993) Vol. 61, pp. 1-23. Advances in Cancer Research.
Publisher: Academic Press, Inc. 1250 Sixth Ave., San Diego, California 92101, USA.
ISSN: 0065-230X. ISBN: 0-12-006661-0.
DOCUMENT TYPE: General Review
LANGUAGE: English

L7 ANSWER 11 OF 11 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 90199728 MEDLINE
DOCUMENT NUMBER: 90199728 PubMed ID: 2138505
TITLE: Inhibition of transformation in cultured rat tracheal epithelial cells by potential chemopreventive agents.
AUTHOR: Steele V E; Kelloff G J; Wilkinson B P; Arnold J T
CORPORATE SOURCE: Environmental Sciences Division, NSI Technology Services Corporation, Research Triangle Park, North Carolina 27709.
CONTRACT NUMBER: N01-CN55503-03 (NCI)
SOURCE: CANCER RESEARCH, (1990 Apr 1) 50 (7) 2068-74.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199004
ENTRY DATE: Entered STN: 19900601
Last Updated on STN: 19900601
Entered Medline: 19900430

AB Twenty-eight compounds were screened for chemopreventive activity by using a rat tracheal epithelial cell transformation inhibition assay. In this new assay, chemicals were tested for their ability to inhibit the formation of transformed rat tracheal epithelial cell colonies which arise following exposure to the carcinogen benzo(a)pyrene. The 15 positive compounds were ***N*** - ***acetylcysteine***, bismuththiol, calcium glucarate, (+/-) catechin, diallyl disulfide, glycaric acid, D-glucaro-1,4-lactone, N-(4-hydroxyphenyl) ***retinamide***, D-limonene, mesna, ***retinoic*** ***acid***, rutin, quercetin, silymarin, and taurine. In examining the nature of compounds that inhibited rat tracheal epithelial cell transformation, several possible chemopreventive mechanisms appeared to be predominant: compounds that were positive (a) increased glutathione levels or enhanced conjugation; (b) increased cytochrome P-450 activity; (c) displayed nucleophilic activity; or (d) induced differentiation. Thirteen compounds were negative in the rat tracheal epithelial transformation inhibition assay: crocetin, difluoromethylornithine, ellagic acid, esculetin, enoxalone, ibuprofen, levamisole, nordihydroguaiaretic acid, L-2-oxothiazolidine-4-carboxylate, piroxicam, sodium butyrate, D-alpha- ***tocopherol*** acetate, and polyethylene glycol 400. It was evident from these results that this assay would not detect compounds that were (a) anti-promoting in nature; (b) glutathione inhibitors; (c) differentiation inhibitors; (d) O6-methylguanine inhibitors; (e) organ specific; or (f) inactive. The rat tracheal epithelial cell transformation inhibition assay appeared to identify chemopreventive compounds that act at early stages of the carcinogenic process.

=> d his

(FILE 'HOME' ENTERED AT 19:11:34 ON 23 JAN 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 19:11:57 ON 23 JAN 2003

L1 165660 S RETINOID OR RETINOL OR RETINAMIDE OR (RETINOIC ACID)
L2 17009 S N-ACETYLCYSTEINE
L3 273934 S CHOLESCIFEROL OR (VITAMIN K) OR TOCOPHEROL OR (ASCORBIC ACID)
L4 119 S L1 (P) L2
L5 696 S L2 (P) L3
L6 19 S L4 (P) L3
L7 11 DUPLICATE REMOVE L6 (8 DUPLICATES REMOVED)

=> s niacin or thiamine or riboflavin or (folic acid) or pyrodoxine or (pantothenic acid) or niac
L8 154568 NIACIN OR THIAMINE OR RIBOFLAVIN OR (FOLIC ACID) OR PYRODOXINE
OR (PANTOTHENIC ACID) OR NIACINAMIDE

=> s (lipoic acid) or (dihydrolipoic acid) or (amino acid)
4 FILES SEARCHED...

L9 1958819 (LIPOIC ACID) OR (DIHYDROLIPOIC ACID) OR (AMINO ACID)

=> s l8 or l9

L10 2103109 L8 OR L9

=> s l7 (p) l10

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L71 (P) L61'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L73 (P) L62'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L75 (P) L63'
L11 3 L7 (P) L10

=> d l11 1-3 ibib abs

L11 ANSWER 1 OF 3 MEDLINE

ACCESSION NUMBER: 2000126354 MEDLINE
DOCUMENT NUMBER: 20126354 Pub ID: 10657911
TITLE: Lung cancer chemoprevention.
AUTHOR: Khuri F R; Lippman S M
CORPORATE SOURCE: Departments of Thoracic/Head and Neck Medical Oncology, The University of Texas M. D. Anderson Cancer Center, Houston, Texas 77030, USA.
SOURCE: SEMINARS IN SURGICAL ONCOLOGY, (2000 Mar) 18 (2) 100-5.
Ref: 46
Journal code: 8503713. ISSN: 8756-0437.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000320
Last Updated on STN: 20000320
Entered Medline: 20000307

AB Lung cancer is the leading cause of cancer death in the United States. The persisting grim lung cancer incidence and mortality figures argue powerfully for new approaches such as chemoprevention for controlling this disease. ***Retinoids*** are among the most intensively studied cancer chemoprevention agents, including in the lung. Several randomized clinical or translational chemoprevention trials (e.g., of ***retinoids***, beta-carotene, or combined ***folic*** ***acid*** and vitamin B(12)) have been conducted in lung pre-malignancy. ***Retinoid*** studies have produced important data on molecular/cellular markers of lung carcinogenesis, e.g., loss of heterozygosity (LOH) at 3p and 9p and ***retinoic*** ***acid*** receptor-beta (RAR-beta). Two large randomized trials with a lung cancer endpoint, the Alpha-Tocopherol, Beta-Carotene (ATBC) Prevention Study and the Beta-Carotene and ***Retinol*** Efficacy Trial (CARET), found that beta-carotene (+/- ***retinol***) was harmful (in smokers). Recently completed lung-second-primary-tumor-prevention trials include the ***retinoids*** retinyl palmitate and 13-cis- ***retinoic*** ***acid*** (13cRA) and ***N*** - ***acetylcysteine*** (NAC). Vitamin E and selenium show promise for lung cancer prevention, based on positive secondary/subset analyses of three large-scale, randomized National Cancer Institute (NCI) cancer prevention trials. Future directions of lung cancer chemoprevention include the study of molecular markers of risk and drug activity, molecular targeting study, improved imaging techniques (e.g., molecular imaging) and new drug delivery systems.
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L11 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:55925 BIOSIS
DOCUMENT NUMBER: PREV199800055925
TITLE: Chemoprevention of aerodigestive cancer.
AUTHOR(S): Berwick, Marianne (1); Schantz, Stimson
CORPORATE SOURCE: (1) Dep. Epidemiology Biostatistics, Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021 USA
SOURCE: Cancer and Metastasis Reviews, (1997) Vol. 16, No. 3, pp. 329-347.
ISSN: 0167-7659.
DOCUMENT TYPE: General Review
LANGUAGE: English

L11 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1993:421810 BIOSIS
DOCUMENT NUMBER: PREV199345069435
TITLE: Cancer prevention research trials.
AUTHOR(S): Greenwald, Peter (1); Malone, Winfred F. (1); Cerny, Mary E.; Stern, Harriet R.
CORPORATE SOURCE: (1) Div. Cancer Prevention and Control, Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20892 USA
SOURCE: Vande Woude, G. F. [Editor]; Klein, G. [Editor]. Advances in Cancer Research, (1993) Vol. 61, pp: 1-23. Advances in Cancer Research.

Publisher: Academic Press, Inc. 1250 Sixth Ave. San Diego,
California 92101 USA.

ISSN: 0065-230X. ISBN: 0-12-006661-0.

DOCUMENT TYPE:

General Review

LANGUAGE:

English

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L4 119 S L1 (P) L2
L5 696 S L2 (P) L3
L6 19 S L4 (P) L3
L7 11 DUPLICATE REMOVE L6 (8 DUPLICATES REMOVED)
L8 154568 S NIACIN OR THIAMINE OR RIBOFLAVIN OR (FOLIC ACID) OR PYRODOXIN
L9 1958819 S (LIPOIC ACID) OR (DIHYDROLIPOIC ACID) OR (AMINO ACID)
L10 2103109 S L8 OR L9
L11 3 S L7 (P) L10

=> s l7 (p) composition

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L84 (P) COMPOSITI'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L86 (P) COMPOSITI'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L88 (P) COMPOSITI'
L12 1 L7 (P) COMPOSITION

=> d l12 1 ibib abs

L12 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2000:839120 CAPLUS

DOCUMENT NUMBER: 134:21446

TITLE: Compositions for stabilizing oxygen-labile
pharmaceuticals

INVENTOR(S): Kung, John; Liu, Jue-chen

PATENT ASSIGNEE(S): Johnson & Johnson Consumer Companies, Inc., USA

SOURCE: Eur. Pat. Appl., 17 pp.

CODEN: EPXXDW

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1055720	A2	20001129	EP 2000-304519	20000526
EP 1055720	A3	20010307		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
CA 2309520	AA	20001128	CA 2000-2309520	20000525
JP 2001011441	A2	20010116	JP 2000-158635	20000529
CN 1284327	A	20010221	CN 2000-118833	20000529
BR 2000003780	A	20010403	BR 2000-3780	20000616
US 2002123460	A1	20020905	US 2001-33492	20011227
PRIORITY APPLN. INFO.:			US 1999-136442P	P 19990528
			US 1999-361425	A 19990727

AB This invention relates to ***compsns*** and methods for stabilizing oxygen-labile species. More particularly, it relates to ***compsns*** contg. 1 or more oil- and/or water-sol. oxygen-labile species and one or more stabilizing elements. It also relates to methods of making such ***compsns*** and methods of using such ***compsns***. Thus, a formulation contained water 73.96, disodium EDTA 0.20, phenoxyethanol 0.73, methylparaben 0.20, propylparaben 0.07 and hydroxyethyl cellulose 1.00% for the water phase; BHT 0.10, GMS 2.00 cetearyl glucoside 3.000, C12-15 alkyl benzoate 2.00, avobenzone 2.00, octyl methoxycinnamate 4.00,

and ascorbyl palmitate 0.50% for the oil phase; ***ascorbic***
acid 5.00, ***tocopherol*** 0.05, ***retinol*** 0.25,
lactoferrin and thioxanthine and uric acid 1.00, ***N*** -
acetylcysteine 0.01, EtOH 2.78 and 20% NaOH 9.04% as the
additives. After a 13-wk incubation at 40.degree., 90% vitamin C and 96%
vitamin A remained in the ***compn***

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FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
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L7 11 DUPLICATE REMOVE L6 (8 DUPLICATES REMOVED)
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L9 1958819 S (LIPOIC ACID) OR (DIHYDROLIPOIC ACID) OR (AMINO ACID)
L10 2103109 S L8 OR L9
L11 3 S L7 (P) L10
L12 1 S L7 (P) COMPOSITION

=> s humectant or antioxidant or preservative or fragrance or (surface active agent) or binder or
L13 552432 HUMECTANT OR ANTIOXIDANT OR PRESERVATIVE OR FRAGRANCE OR (SURFAC
E ACTIVE AGENT) OR BINDER OR (SKIN PROTECTING AGENT)

=> s l7 and l13

L14 2 L7 AND L13

=> d l14 1-2 ibib abs

L14 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2001:482634 CAPLUS

DOCUMENT NUMBER: 135:298715

TITLE: Effects of ***antioxidant*** vitamins on
anti-IgE-induced mediator release from human basophils
AUTHOR(S): Strenzke, N.; Grabbe, J.; Plath, K. E. S.; Wolff, H.
H.; Gibbs, B. F.

CORPORATE SOURCE: Department of Dermatology, Medical University of
Lubeck, Lubeck, D-23538, Germany

SOURCE: Inflammation Research (2001), 50(Suppl. 2), S49-S50
CODEN: INREFB; ISSN: 1023-3830

PUBLISHER: Birkhaeuser Verlag

DOCUMENT TYPE: Journal

LANGUAGE: English

AB It was examd. whether ***antioxidants*** may provide beneficial
effects by inhibiting IgE-dependent basophil mediator release.
Ascorbic ***acid***, ***N*** - ***acetylcysteine*** and
glutathione were dissolved in RPMI 1640 medium and the pH was cor. to 7.4.
Glutathione and its precursor ***N*** - ***acetylcysteine*** had no
influence on IgE-dependent histamine release from human basophils, while
ambroxol was shown to be a highly efficacious inhibitor. All the vitamins
tested showed no effect on histamine release in the absence of anti-IgE.
In the histamine release expts., ambroxol was the only effective inhibitor
of interleukin (IL)-4 and IL-13 releases. Compared to solvent controls,
N - ***acetylcysteine***, ***retinoic*** ***acid*** and
.beta.-carotene did not considerably alter anti-IgE induced cytokine
releases. ***Ascorbic*** ***acid*** strongly enhanced IL-13
release, which was not significant because of the donor variation in IL-13
release. A major role for reactive oxygen species in the IgE-dependent
activation of human basophils was not supported by the resulting data.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1998:497254 CAPLUS

DOCUMENT NUMBER: 129:230153